Central Sensitisation & Medical Misogyny 101

Pain affects over 20% of the global population, creating significant health and economic burdens.

This presentation explores the journey from acute to chronic pain, focusing on central sensitisation mechanisms but central sensitisation does not touch all humanity equally and I will present my thoughts on this.

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The opinions expressed are my own! And not that of my employer. Manchester Foundation Trust.

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What is Pain? Defining the Experience

Sensory Component

🔥 Nociceptive signals transmit location and intensity information.

Chronic Pain Burden

⏱️ Persistent pain (>3 months) leads to disability, depression, and decreased quality of life, affecting 20% of adults worldwide.

Clinical Significance

🏥 Primary reason patients seek care, with 10-40% global prevalence.

Cognitive Component

🧠 Brain interprets and contextualises pain signals.

Emotional Component

😣 Pain evokes distress, anxiety, and suffering.

Social Component

👥 Cultural factors and interpersonal relationships influence perception.

Defence Mechanism

🛡️ Acts as protection against noxious stimuli and secondary insults.

Categories of Pain: Understanding the Differences

Nociceptive Pain

Protective system response to noxious stimuli

- Most frequent type of pain
- Transient response to harmful factors
- Examples: cutting, burn injuries

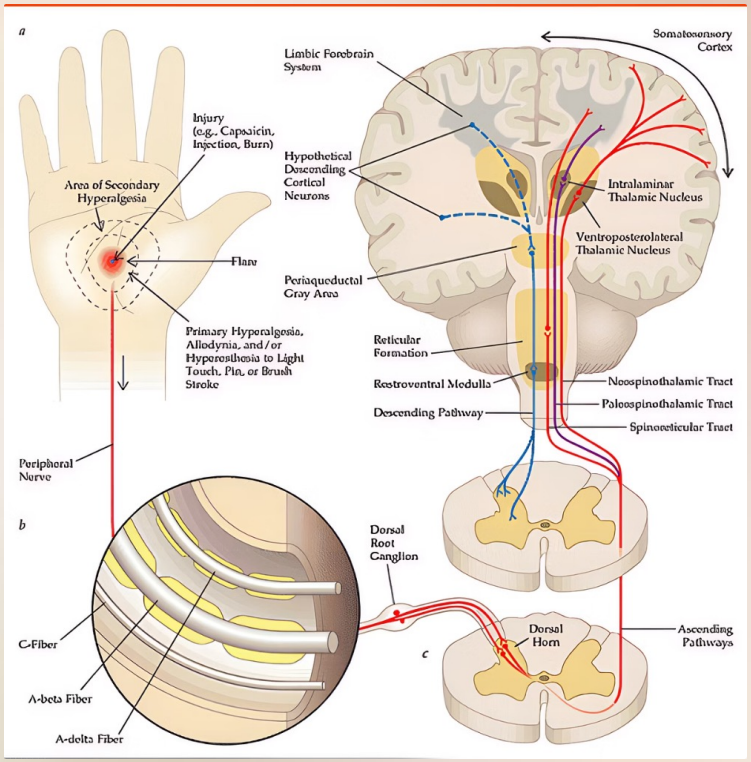
Neuropathic Pain

Arises from somatosensory system lesions

- Affects 7-10% of general population
- Represents 20-25% of chronic pain cases
- Often resistant to standard analgesics

Nociplastic Pain

- Altered nociception without clear tissue damage
- Affects 5-15% of general population
- Shows significant female predominance
- Influenced by genetic and psychosocial factors



Basic Circuits of Pain: The Transmission Pathway

Nociceptors

Peripheral transducers located in skin, mucosa, and organs detect thermal, mechanical, and chemical stimuli.

Nerve Fibres

Aδ fibres transmit sharp, fast pain while C fibres carry burning, slower pain signals.

Spinal Cord Processing

Signals reach dorsal horn where projection neurons integrate and transmit to the brain.

Brain Processing

The "pain matrix" includes thalamus, somatosensory cortex, anterior cingulate cortex, and insula, interpreting signals as pain experience.

The Gate Control Theory

> "Pain is not simply a direct product of activation of nociceptive afferents, but is regulated by interactions between different neurons and circuits in the CNS." - Melzack & Wall, 1965

1. **Primary Afferent Input**

   Large-diameter fibres inhibit and small-diameter fibres facilitate transmission of pain signals

2. **Substantia Gelatinosa "Gate"**

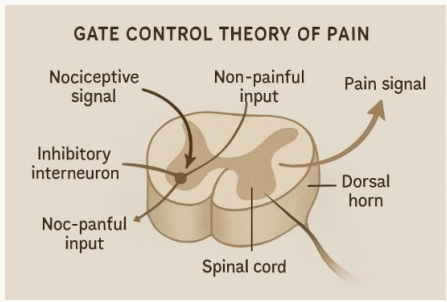
   Modulates transmission of afferent signals to projection neurons through presynaptic inhibition

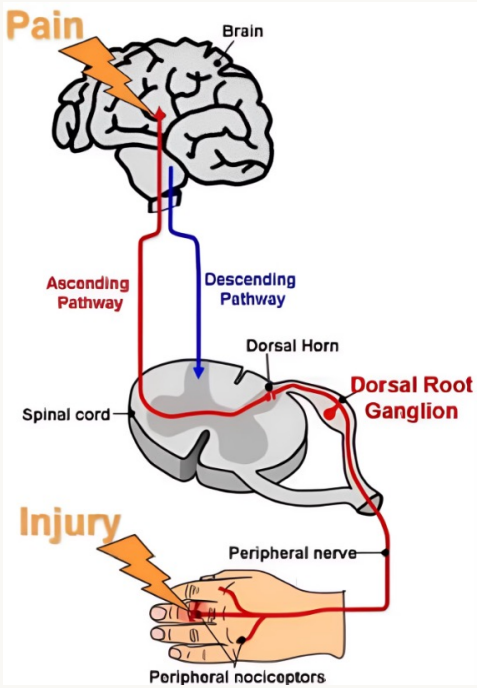
3. **Central Control**

   Descending pathways from brain centres can close or open the gate, explaining pain modulation by psychological factors

4. **Projection to Action System**

   When gate allows sufficient activity, the transmission cells activate neural mechanisms for pain perception and response





The Transition: From Acute to Chronic Pain

Initial Injury

Tissue damage activates nociceptors, sending signals via A-delta and C fibres.

2

Inflammatory Response

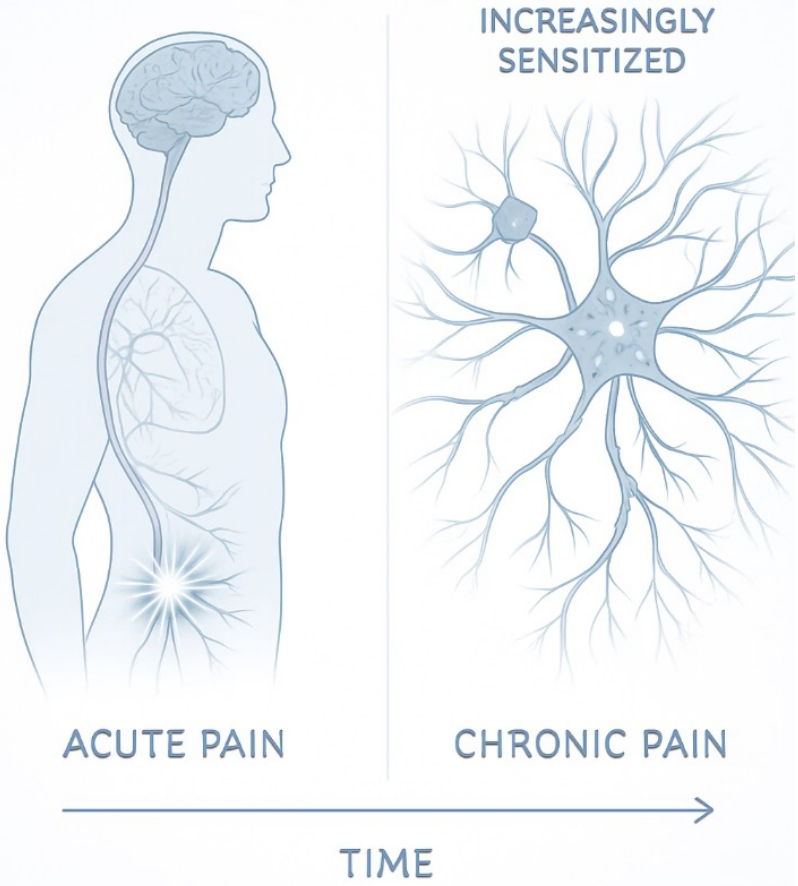
Release of inflammatory mediators sensitises peripheral nerves.

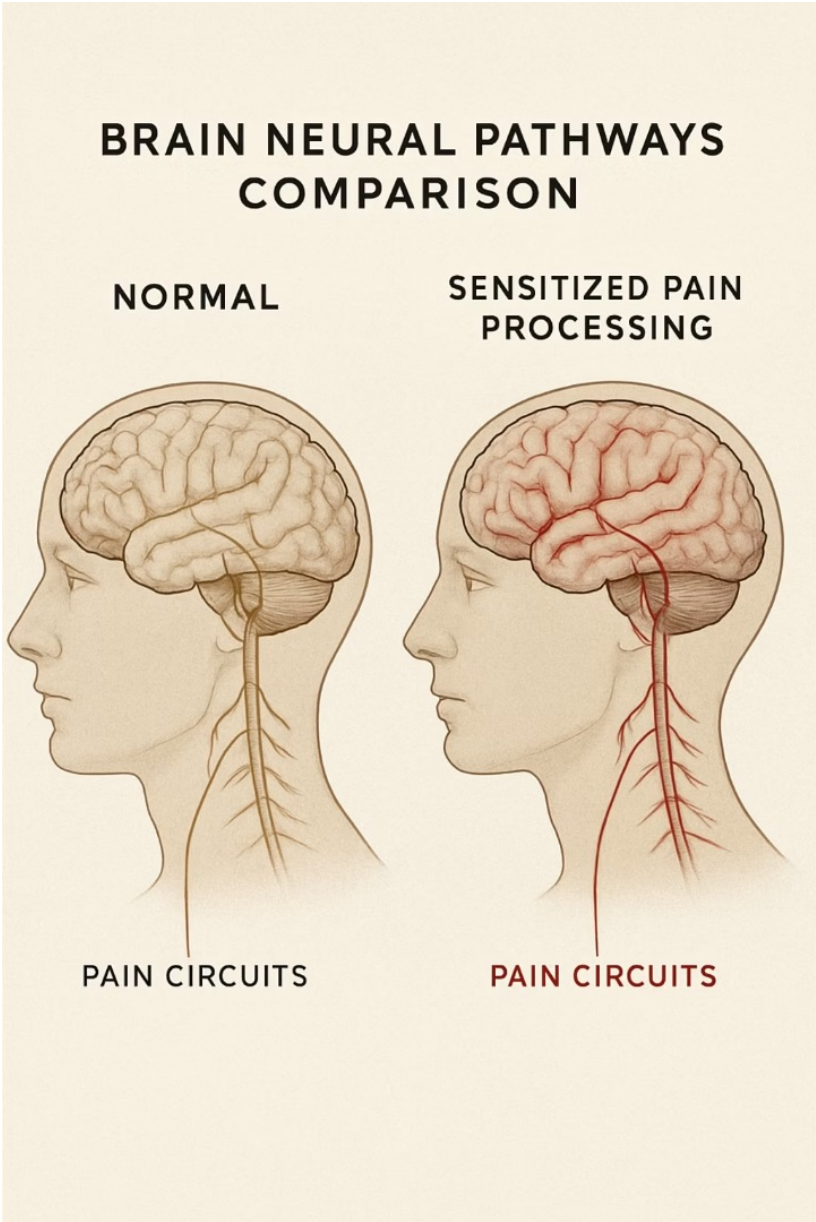
Central Sensitisation

Persistent input enhances synaptic efficacy in pain pathways.

Chronification

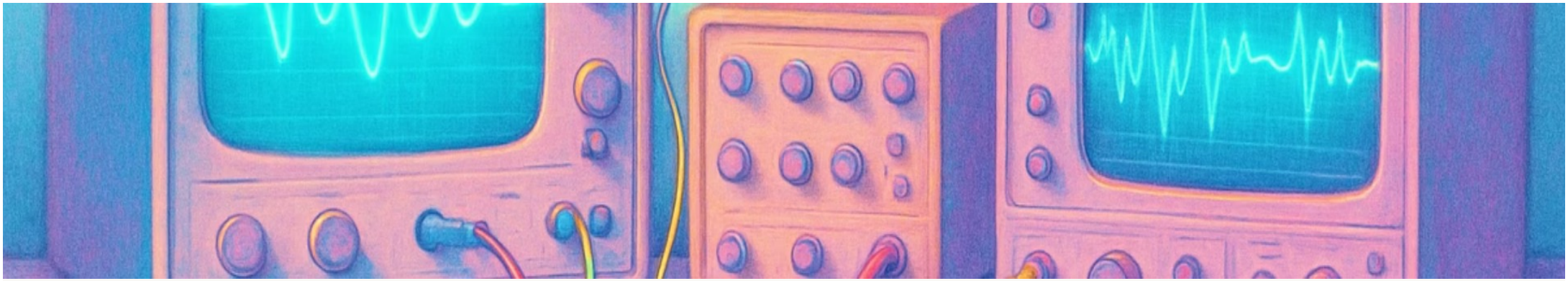
Neural plasticity creates self-sustaining pain circuits independent of initial trigger.





The Fundamental Shift in Pain Processing

Normal Pain Processing	Centrally Sensitised State
High-threshold nociceptors required	Low-threshold inputs can trigger pain
Pain directly relates to stimulus intensity	Pain disproportionate to stimulus
Pain confined to injury area	Pain spreads beyond original site
Pain stops when stimulus ends	Pain persists after stimulus removal
Pain serves protective function	Pain becomes a pathological state



The Discovery of Central Sensitisation

1983: First Evidence

1

Woolf demonstrated that noxious heat stimuli could produce long-lasting changes in the excitability of flexor motor neurons, with reduced thresholds and enlarged receptive fields.

Key Experiments

2

Three experiments showed these changes were due to alterations in the CNS: A-β fiber activation of motor neurons after conditioning, persistence despite local anesthetic block, and mimicking by brief C-fiber electrical stimulation.

Expanded Understanding

3

Similar changes were soon described in lamina I and V neurons in the dorsal horn, spinal nucleus pars caudalis, thalamus, amygdala, and anterior cingulate cortex.

Modern Imaging Confirmation

4

fMRI, PET, and magnetoencephalography have revealed in humans that multiple brain structures exhibit changes compatible with central sensitisation.

Understanding Normal vs. Sensitised Pain Pathways

Normal Pain Pathway

Acute nociceptive pain is the physiological sensation of hurt resulting from activation of nociceptive pathways by peripheral stimuli of sufficient intensity to lead to or threaten tissue damage (noxious stimuli).

Nociception, the detection of noxious stimuli, is a protective process that helps prevent injury by generating both a reflex withdrawal and an unpleasant sensation.

Sensitised Pain Pathway

In central sensitisation, pain is no longer coupled to the presence, intensity, or duration of noxious peripheral stimuli. Instead, it represents an abnormal state of responsiveness in the nociceptive system.

The pain is generated as a consequence of changes within the CNS that alter how it responds to sensory inputs, rather than reflecting peripheral noxious stimuli.

From Acute to Chronic Pain: The Time Course

1. **Acute Phase (0-7 days)**

   Tissue damage activates nociceptors. Inflammation begins with cytokine release. Pain directly relates to injury severity.

2. **Subacute Phase (1-6 weeks)**

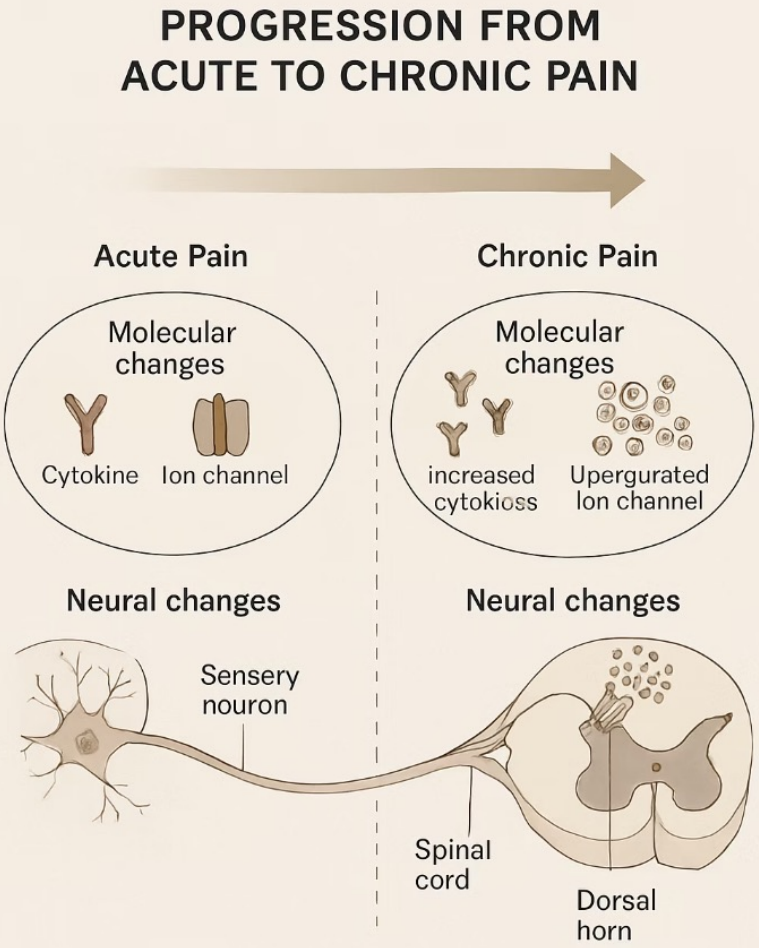
   Early central changes emerge. Synaptic potentiation strengthens pain pathways. Gene expression changes begin.

3. **Transition Phase (6-12 weeks)**

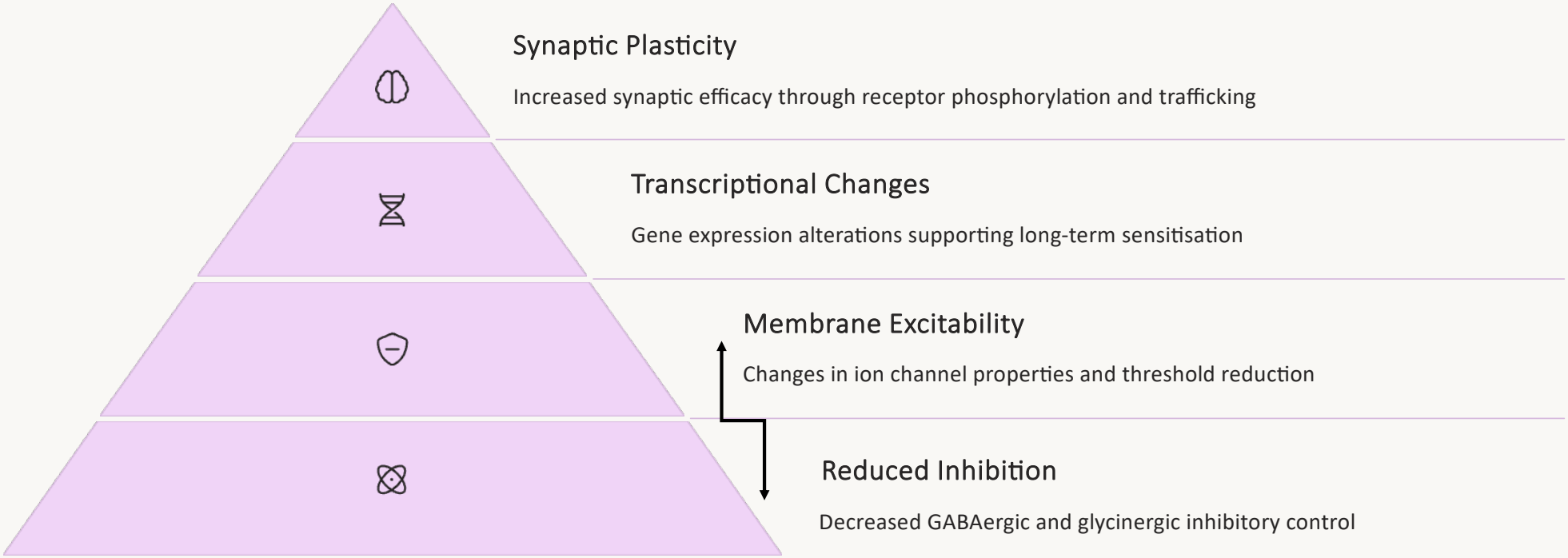
   Critical period for chronification. Structural and functional neuroplasticity occurs. Psychosocial factors gain importance.

4. **Chronic Phase (>12 weeks)**

   Self-sustaining pain circuits established. Widespread central sensitisation present. Pain persists independent of initial trigger.



Mechanisms of Central Sensitisation



Central sensitisation comprises two temporal phases:
an early phosphorylation-dependent, transcription-independent phase resulting from rapid changes in glutamate receptor and ion channel properties
a later transcription-dependent phase driving synthesis of new proteins responsible for longer-lasting forms of central sensitisation observed in pathological conditions.

Mediators of Central Sensitisation

Glutamate Receptors

NMDA, AMPA, and metabotropic glutamate receptors are critical for initiating and maintaining central sensitisation. NMDA receptor activation is essential, as its blockade prevents and reverses hyperexcitability of nociceptive neurons.

Neuropeptides

Substance P and CGRP, co-released with glutamate by unmyelinated peptidergic nociceptors, cause long-lasting membrane depolarization and contribute to temporal summation of C-fiber-evoked synaptic potentials.

Neurotrophic Factors

BDNF, synthesized by nociceptor neurons and released into the spinal cord in an activity-dependent manner, enhances NMDA receptor-mediated responses and activates several signaling pathways in spinothalamic tract neurons.

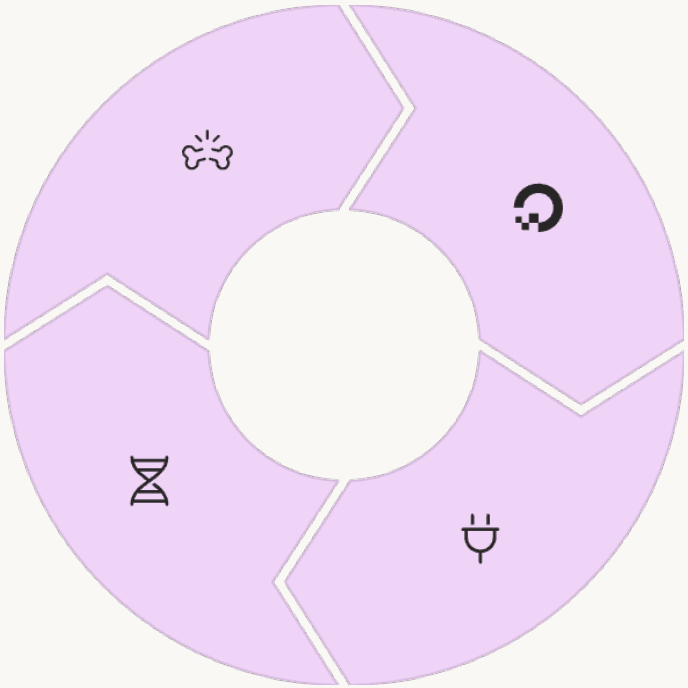
Inflammatory Mediators

Bradykinin, produced in the spinal cord in response to intense peripheral noxious stimuli, boosts synaptic strength by activating PKC, PKA, and ERK pathways.

Signaling Pathways in Central Sensitisation

Calcium Influx

Increased intracellular calcium through NMDA receptors, calcium-permeable AMPA receptors, and voltage-gated calcium channels triggers sensitization



Kinase Activation

PKC, PKA, CaMKII and ERK phosphorylate receptors and ion channels, altering their properties

Transcriptional Changes

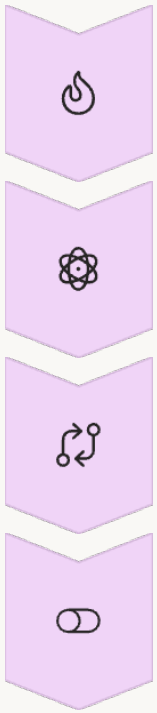
CREB activation drives expression of genes including c-Fos, NK1, TrkB, and Cox-2

Receptor Modification

Phosphorylation changes receptor trafficking, threshold, and kinetics, enhancing synaptic efficacy

An increase in intracellular calcium beyond a certain threshold is the key trigger for initiating activity-dependent central sensitisation. This calcium influx activates multiple intracellular kinases that phosphorylate ionotropic NMDA and AMPA glutamate receptors, changing their activity and trafficking to or from the membrane, which produces the functional changes that manifest as central sensitization.

Central Sensitization in Inflammatory Pain



Peripheral Inflammation

Triggers phenotypic switch in primary sensory neurons, changing their neurochemical character due to alterations in transcription and translation

Neuropeptide Expression

Large DRG neurons begin to express substance P and BDNF, enabling myelinated fibers to generate central sensitization

COX-2 Induction

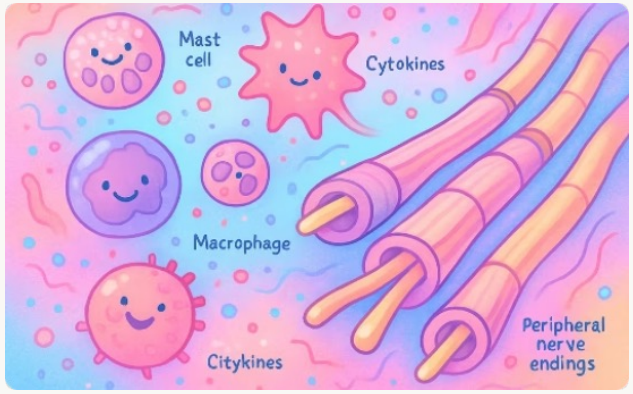
Drives production of prostaglandin E2 (PGE2), which potentiates AMPA and NMDA receptor currents

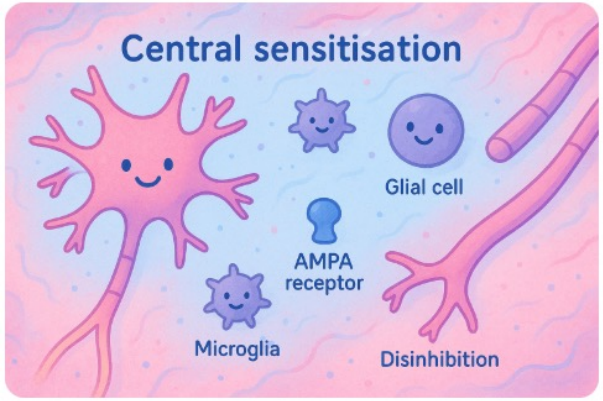
AMPA Receptor Switch

Shift from GluR2/3 to GluR1-containing AMPARs, allowing calcium influx that further drives sensitization

Peripheral inflammation induces a critical pathway involving cyclooxygenase-2 (COX-2) induction in dorsal horn neurons, driving production of prostaglandin E2. PGE2 binds to EP2 receptors to potentiate AMPA and NMDA receptor currents, activate nonselective cation channels, and reduce inhibitory glycinergic neurotransmission, collectively enhancing pain sensitivity.

Central Sensitisation in Inflammatory Pain





Peripheral Mechanisms

Inflammation induces a phenotypic switch in sensory neurones.

- NGF drives expression of substance P and BDNF
- Large DRG neurones begin expressing pain mediators
- Increased excitability in primary afferents

Central Mechanisms

Spinal cord undergoes significant plasticity.

- COX-2 induction drives PGE2 production
- AMPA receptor subunit switch to GluR1-containing
- Calcium-permeable AMPARs increase calcium influx
- Reduced inhibitory control in dorsal horn

Central Sensitization in Neuropathic Pain

Nerve Injury

Damaged and non-damaged A and C-fibers generate spontaneous action potentials (ectopic input)

Glial Activation

Massive activation of microglia and astrocytes, releasing cytokines, BDNF, and other factors

Disinhibition

Reduction in GABAergic and glycinergic inhibitory currents through multiple mechanisms

Structural Changes

A-β fibers sprout from deeper to superficial laminae, making contact with nociceptive neurons

After peripheral nerve injury, damaged and non-damaged sensory neurons exhibit massive transcriptional changes altering their membrane properties, growth, and transmitter function. Glial cells play an essential role in triggering central sensitization through their interaction with neurons, releasing trophic factors, neurotransmitters, cytokines, and reactive oxygen species that increase neuronal excitability and reduce inhibition.

Manifestations of Central Sensitisation

Spontaneous Pain

Pain occurs without peripheral stimulation.

Allodynia

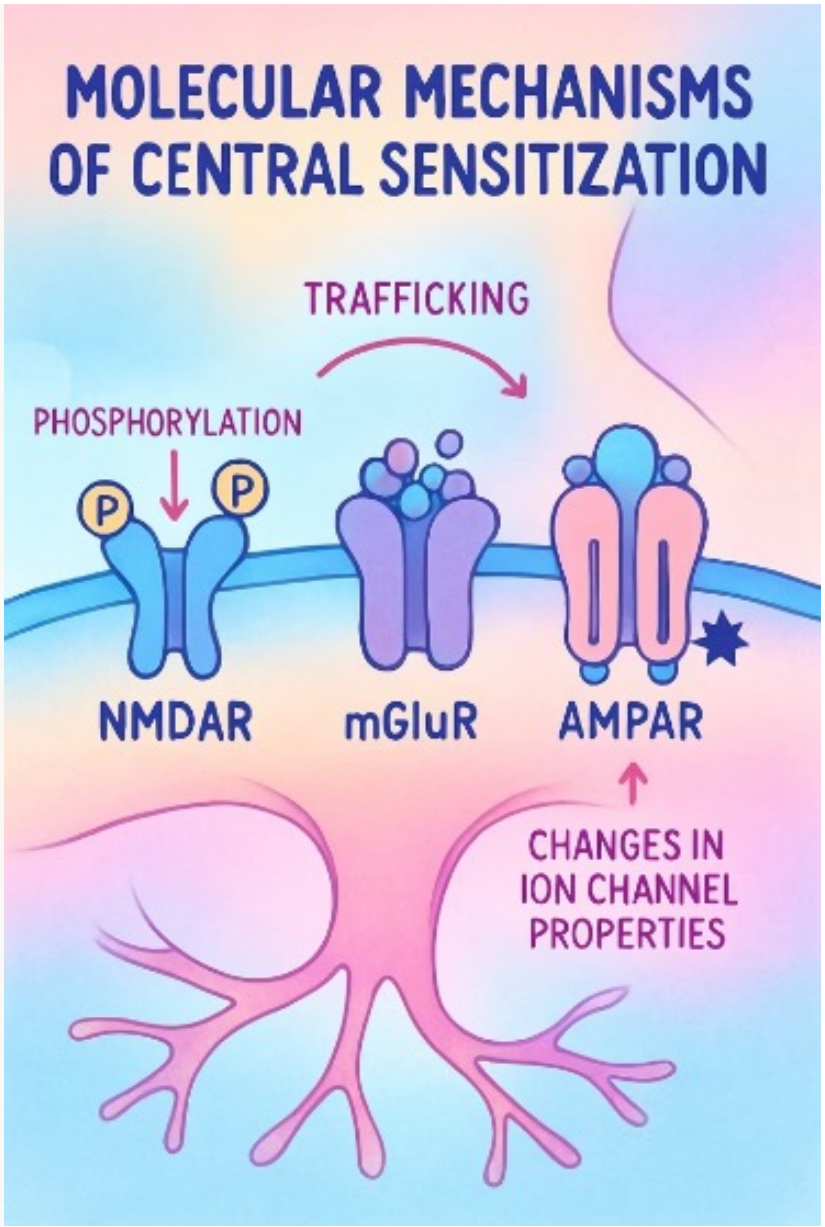
Normally painless stimuli become painful.

Hyperalgesia

Increased pain response to normally painful stimuli.

Expanded Receptive Fields

Pain spreads beyond the original injury site.



Central Sensitisation in Neuropathic Pain

1000+

Gene Expression Changes

Transcripts altered in DRG neurones after nerve injury

24/7

Spontaneous Activity

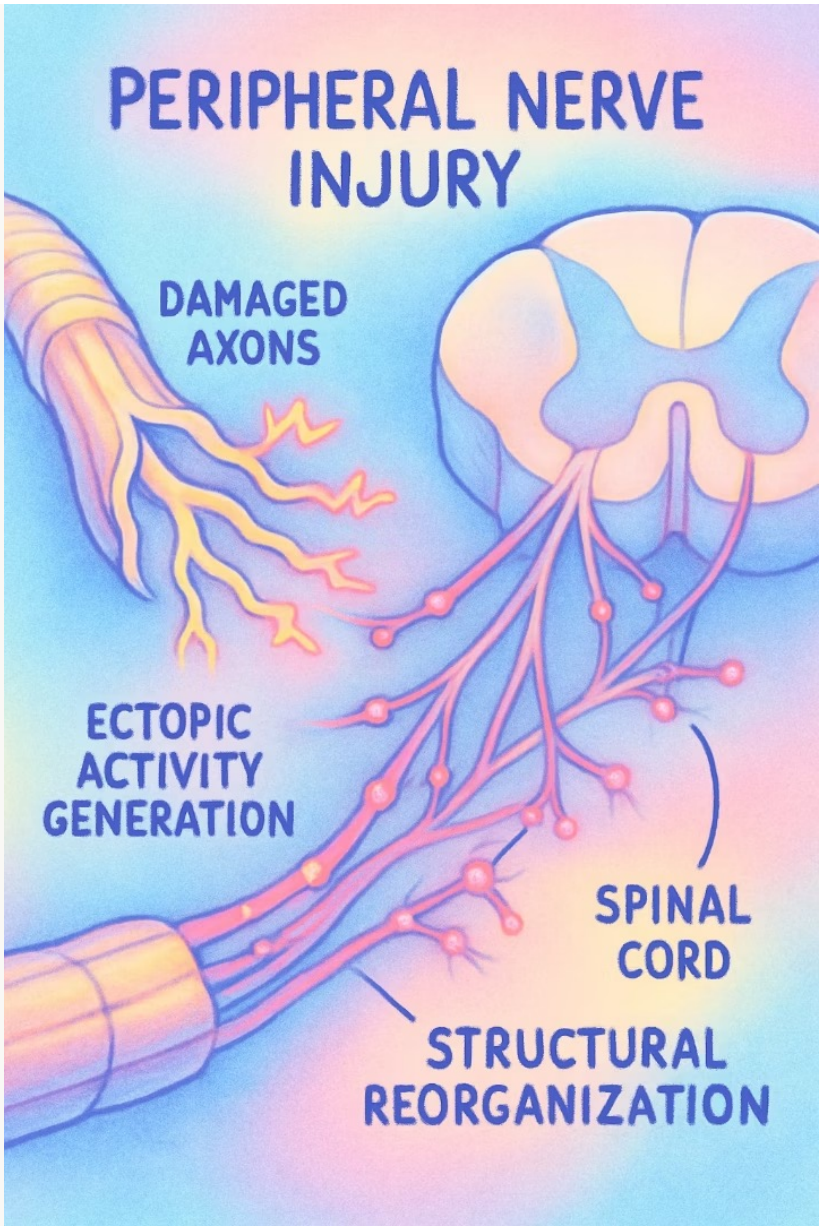
Continuous ectopic firing in damaged nerves

70%

Inhibitory Loss

Approximate reduction in inhibitory control

After peripheral nerve injury, damaged and non-damaged A- and C-fibres begin to generate spontaneous action potentials. This ectopic input triggers massive transcriptional changes, much greater than those seen in inflammation.



Glial Involvement in Central Sensitisation

Glia, microglia, oligodendrocytes, schwann cells, satellite glial cells

Microglial Activation

Nerve injury rapidly activates microglia in the dorsal horn

- Release pro-inflammatory cytokines (IL-1β, TNF-α)
- Produce BDNF, downregulating KCC2
- Show clear sex differences in pain modulation

Astrocyte Contributions

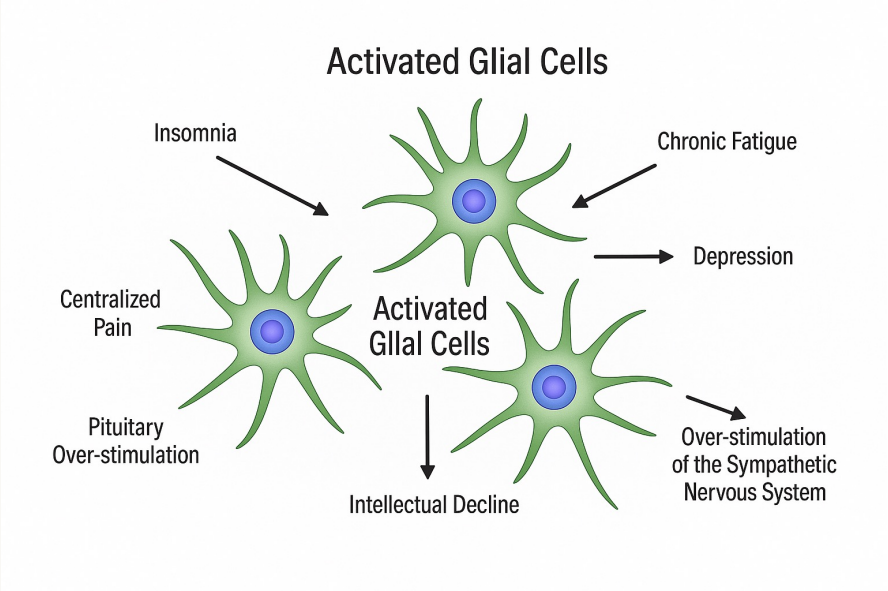
Activated later but produce more persistent effects

- Release cytokines and chemokines
- Reduce glutamate uptake, increasing excitation
- Form "tetrapartite synapses" with neurones

Therapeutic Implications

Targeting glia offers new pain management approaches

- Minocycline inhibits microglial activation
- Propentofylline suppresses astrocyte function
- Sex-specific responses to glial modulators



Glia, the “mum” of the nervous system, support cells doing the unseen work, active regulators of neuronal function, immune defence, and pain modulation.

🧠 The Main Types of Glial Cells

Type	Location	Main Functions	Role in Pain
Astrocytes	Brain & spinal cord	Maintain the chemical environment around neurons; regulate neurotransmitters like glutamate; form part of the blood–brain barrier.	Become “reactive” after injury, releasing cytokines and glutamate → amplify pain signalling and maintain central sensitisation.
Microglia	Brain & spinal cord (resident immune cells)	Detect injury or infection; phagocytose debris; release inflammatory mediators.	Act as the **first responders** to nerve injury — release IL-1β, TNF-α, and BDNF, which enhance excitability of dorsal horn neurons.
Oligodendrocytes	CNS	Form myelin around axons for rapid signal conduction.	Their dysfunction contributes to demyelination and neuropathic pain.
Schwann cells	PNS	Myelinate peripheral nerves; repair after injury.	Release cytokines and growth factors that can sensitize peripheral nociceptors.
Satellite glial cells	Dorsal root ganglia	Envelop sensory neuron cell bodies.	Communicate with neurons via ATP and cytokines → contribute to peripheral sensitisation.

Sex-Based Differences in Pain Processing

Sex-based differences in pain processing present crucial implications for clinical management. These variations arise from distinct neurobiological mechanisms.

Understanding these differences allows for more personalised approaches to pain therapy. Pain experiences vary significantly between sexes due to hormonal and neurological factors.

Sexual Dimorphism in Pain

Prevalence Differences

Women have higher rates of chronic pain conditions:

- Fibromyalgia (7:1 female predominance)
- Irritable bowel syndrome (2:1)
- Migraine (3:1)
- Temporomandibular disorders (2:1)

Physiological Factors

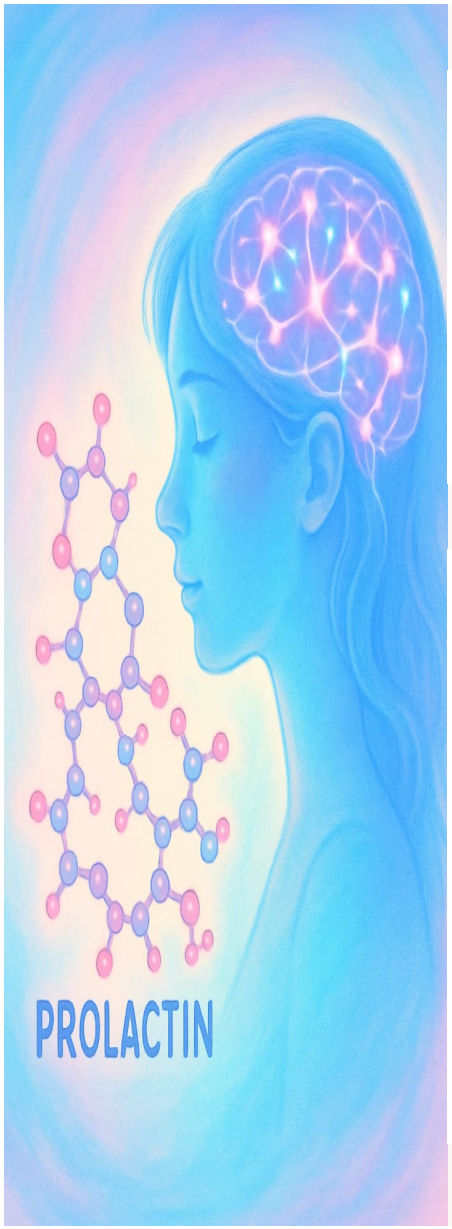
Several mechanisms contribute to sex differences:

- 17β-oestradiol enhances neuronal excitability
- Testosterone has protective effects
- Prolactin receptors crucial for female pain
- Sex-specific immune responses to injury

Brain Connectivity Differences

Neural circuits differ between sexes:

- Female amygdala connects more with prefrontal modulatory regions
- Male amygdala links more with anterior cingulate and insula
- PAG functions show clear sexual dimorphism



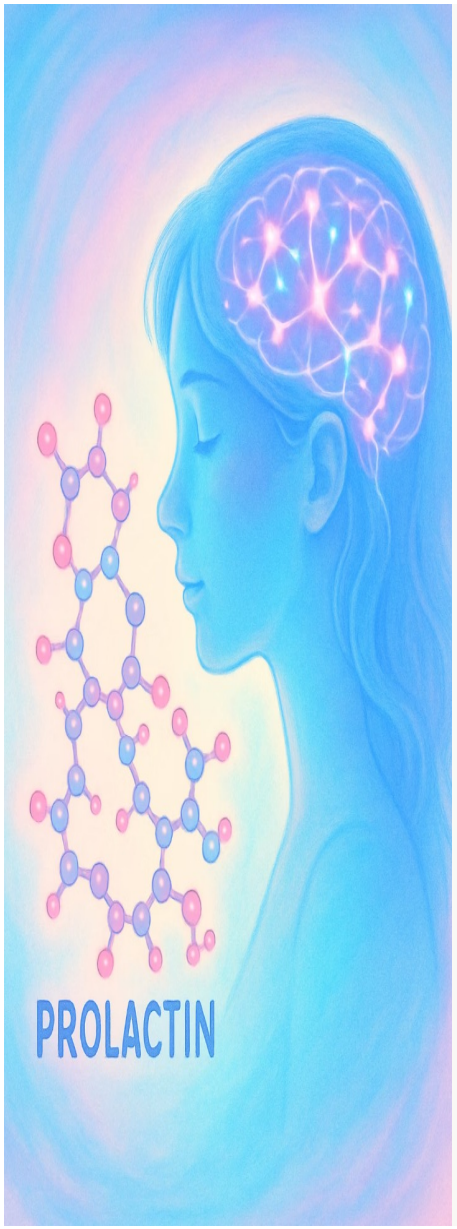
🧬 What Is Prolactin?

Prolactin (PRL) is a **peptide hormone** produced mainly by the **anterior pituitary gland**, under the control of the hypothalamus.

Its name comes from its most famous role — *promoting lactation* — but it actually has **over 300 known biological functions** across reproduction, metabolism, immunity, and the nervous system.

🧠 Where It Comes From

- **Produced by:** lactotroph cells in the anterior pituitary.
- **Controlled by:** the hypothalamus, mainly through **dopamine** (which *inhibits* prolactin release).
- **Stimulated by:**
  - Oestrogen
  - Stress
  - Sleep
  - Suckling (in lactating women)
  - Certain neuropeptides and cytokines



Prolactin and Sex Differences in Pain

Higher Female Prolactin Levels

Females have elevated baseline prolactin. This leads to increased pain sensitivity in multiple pathways.

Receptor Density Differences

Pain neurones show greater prolactin receptor density in females. This amplifies pain signalling cascades.

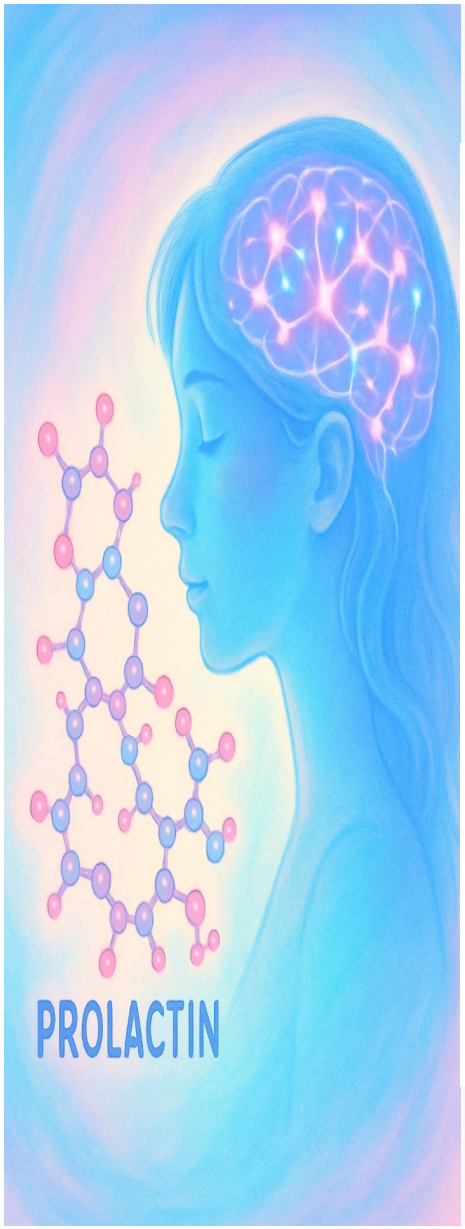
Hormonal Interactions

Oestrogen potentiates prolactin effects. Testosterone provides protective effects by dampening prolactin sensitivity.

Chronic Pain Impact

Prolactin strongly correlates with female chronic pain conditions. This explains higher prevalence rates in females.

But Commissioners, NICE, us doctors, the pharma industry treat us as if we are all the same……



🔥 Prolactin and Pain — an Underappreciated Connection

This is especially relevant to your field (pain medicine and sensitisation biology):

1. **Peripheral sensitisation**
   - Prolactin receptors are expressed on **sensory neurons** (especially in dorsal root ganglia).
   - Elevated prolactin can **enhance nociceptor excitability**, increasing pain perception.
2. **Sex differences in pain**
   - Oestrogen amplifies prolactin release and receptor expression.
   - Animal and human studies show that prolactin-driven sensitisation contributes to **female-predominant pain syndromes** (migraine, fibromyalgia, temporomandibular pain).
   - This may partly explain why certain chronic pain conditions are more common or severe in women.
3. **Central effects**
   - In the spinal cord, prolactin signalling can modulate **glial activation and NMDA receptor sensitivity** — fitting neatly into the central sensitisation framework.

Central Pain Processing Differences: Men vs. Women

Brain Activation Patterns

Women show greater emotional brain activation. The amygdala and insula play prominent roles.

Men demonstrate stronger cognitive-evaluative activation. Their prefrontal cortex shows heightened activity during pain.

Neurotransmitter Systems

Women rely more heavily on opioidergic systems. This affects analgesic efficacy in female patients.

Men exhibit stronger descending inhibition pathways. This may contribute to differing pain tolerance.

Neural Connectivity

Women demonstrate greater limbic-pain area connectivity. This intensifies emotional components of pain.

Men show stronger sensorimotor connectivity. This facilitates physical rather than emotional processing.

Sex Hormone Influences on Pain Perception

Oestrogen Effects

Modulates pain thresholds bidirectionally. Can both enhance and inhibit pain depending on tissue type and concentration.

Hormonal Interactions

Complex interplay affects pain processing. Ratios between hormones may be more important than absolute levels.

Progesterone Influence

Enhances pain inhibition pathways. Contributes to cyclical pain sensitivity throughout menstrual phases.

Testosterone Protection

Provides significant analgesic effects. Higher levels correlate with reduced pain sensitivity in males.

Clinical Implications for Pain Management (Howling in the Gale)

Recognise Sex-Based Differences

Acknowledge biological variations in pain processing. Different mechanisms require different approaches.

Consider Hormonal Status

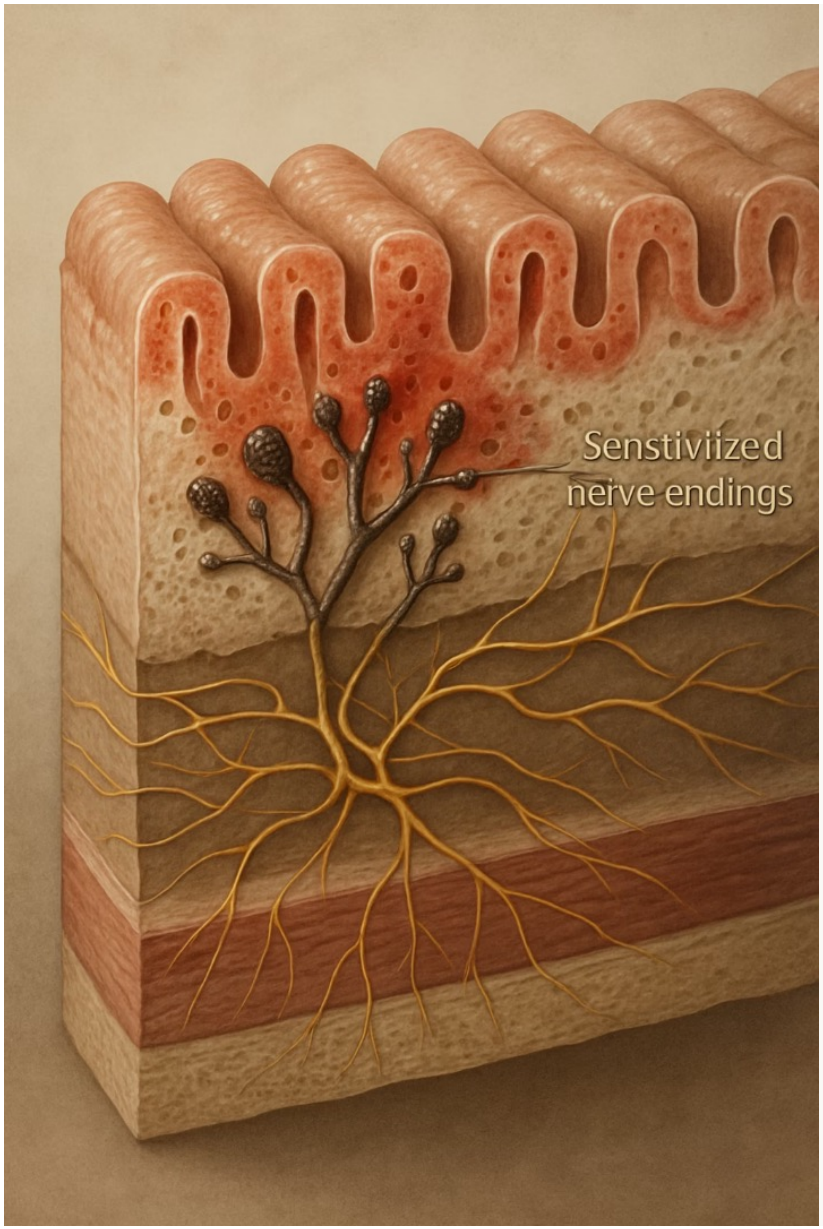
Evaluate menstrual cycle phase in women. Hormonal status affects both pain perception and analgesic efficacy.

Tailor Treatment Approaches

Select medications based on sex-specific mechanisms. Consider hormone-targeting therapies when appropriate.

Implement Personalised Plans

Combine pharmacological and non-pharmacological approaches. Individualise based on patient's unique hormonal profile.



Visceral Hyperalgesia: Understanding the Mechanism

Altered Visceral Sensitivity

Normal sensations from internal organs are perceived as painful.

Peripheral Sensitisation

Inflammatory mediators reduce threshold of visceral afferents.

Central Processing Changes

Spinal and supraspinal circuits amplify incoming signals.

Brain-Gut Axis Dysfunction

Bidirectional communication pathways become dysregulated.

Fibromyalgia: Genetics and Epigenetics Insights

Fibromyalgia (FM) is a complex chronic pain syndrome affecting 5% of the female population & 1% of the male population, characterised by widespread pain persisting for more than three months without obvious organic lesions. Additional symptoms include joint stiffness, fatigue, sleep disturbance, cognitive dysfunction, and depression.

Currently, fibromyalgia diagnosis is based exclusively on clinical assessment according to 2016 ACR criteria, as validated biological biomarkers have not yet been identified. Recent research suggests genetic factors may be responsible for up to 50% of disease susceptibility, while environmental triggers and epigenetic alterations may provide the basis for developing diagnostic biomarkers.

Understanding Fibromyalgia Pathophysiology

Central Sensitisation

Evidence supports central dysregulation at spinal and supra-spinal levels in FM patients compared to controls

Pain Response Abnormalities

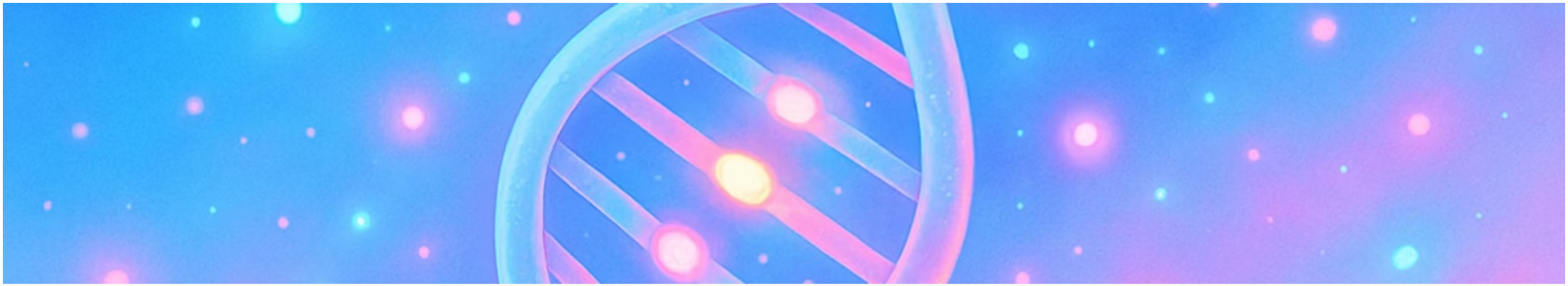
FM patients show exaggerated pain response after sensory stimulation and extended cutaneous silent period

Impaired Pain Modulation

Conditioned pain modulation is consistently reduced or absent in FM subjects, suggesting decreased serotonergic and noradrenergic activities

Altered Neural Processing

Reward/punishment circuit appears impaired, consistent with altered dopaminergic/GABAergic neurotransmission



Genetic Contributions to Fibromyalgia

Familial Risk

Genome-wide linkage scan revealed a 13.6-fold increased risk of developing fibromyalgia in first-degree relatives, strengthening the genetic hypothesis

Candidate Genes

Potential candidate genes found associated with fibromyalgia include SLC64A4 (serotonin transporter), TRPV2, MYT1L (neuronal differentiation), and NRXN3 (nervous system receptor)

SNP Associations

Single nucleotide polymorphisms in genes like COMT, HTR2A, TAAR1, RGS4, CNR1, and GRIA4 have been identified as potentially associated with fibromyalgia susceptibility

Haplotype Influence

Combinations of variants (haplotypes) might affect the risk of fibromyalgia development more than single variants, such as the "high pain sensitivity" ACCG haplotype in the COMT gene

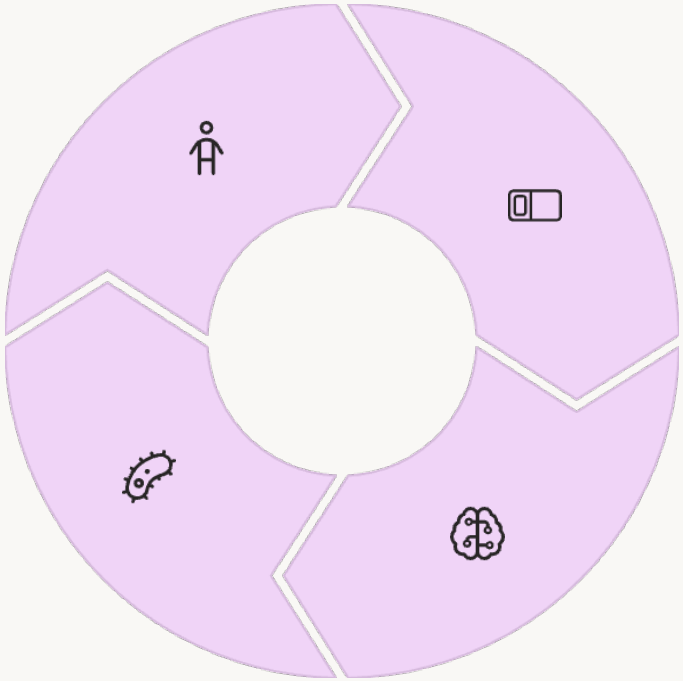
Environmental Influences on Fibromyalgia

Early-Life Events

Physical trauma and psychosocial stressors in early life influence gene expression and contribute to fibromyalgia development

Gut Microbiome

Environment and stress reactions impact gut microbial composition, potentially contributing to mitochondrial dysfunction associated with pain sensitisation



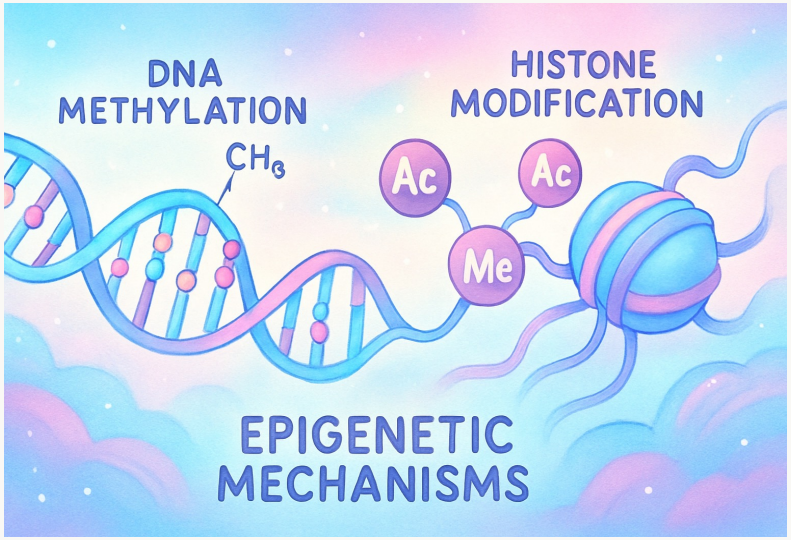
Psychological Stressors

Chronic stress, emotional trauma, and physical assault/abuse are strong predictors of fibromyalgia, particularly in women

HPA Axis Dysfunction

Impairment of hypothalamic-pituitary-adrenal axis leads to inefficient stress response and enhanced sensitivity to pain and fatigue

Epigenetic Mechanisms in Fibromyalgia



DNA Methylation

FM patients show a hypomethylated DNA pattern in genes implicated in stress response, DNA repair, autonomic system response, and subcortical neuronal abnormalities.

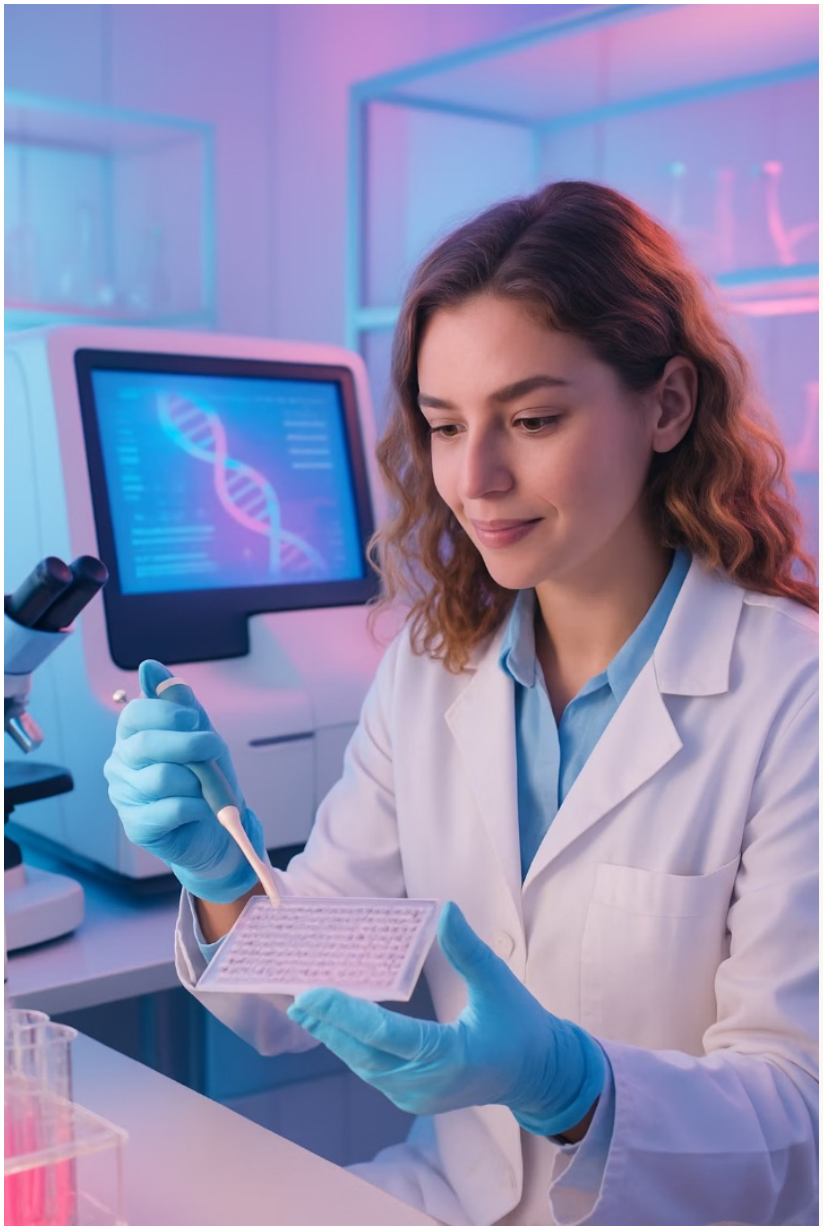
Studies revealed 69 differentially methylated sites in FM cases, with 91% responsible for increased micronuclei frequency.

Genes mapped on differently methylated sites include BDNF, NAT15, HDAC4, PRKCA, RTN1, and PRKG1.

Histone Modifications

Covalent post-translational modifications of histone proteins alter chromatin structure, affecting biological processes including DNA repair, gene transcription, and ageing.

Histone acetylation/deacetylation mechanisms are particularly important in pain conditions. HDAC inhibitors have emerged as potentially implicated in analgesia for both inflammatory and neuropathic pain.



Future Directions for Fibromyalgia Research

Validate Preliminary Findings

Confirm genetic and epigenetic markers in larger multicentre cohorts with precise exclusion criteria and attention to ongoing therapies

Develop Diagnostic Biomarkers

Utilise epigenetic patterns and miRNA profiles from blood samples as accessible biomarkers for objective diagnosis

Explore Epigenetic Treatments

Investigate chromatin-modifying drugs and compounds targeting DNA methylation to potentially reverse aberrant gene expression profiles

Implement Personalised Medicine

Develop targeted therapies based on individual genetic and epigenetic profiles to improve outcomes and reduce side effects

Understanding Fibromyalgia Challenges

2.1M

UK Fibromyalgia Patients

Approximately 3% of the population impacted by this chronic condition

90%

Widespread Pain

Experience debilitating widespread pain and tenderness throughout the body

5-7

Years to Diagnosis

The typical diagnostic journey involves multiple specialists and tests

75%

Mental Health Impact

Experience depression, anxiety and cognitive difficulties often called "fibro fog"

Fibromyalgia creates substantial healthcare burdens. Patients often feel invalidated and dismissed during their diagnostic journey as there are no definitive tests. Traditional pain management approaches frequently fail to address the complex spectrum of symptoms. The Elyfia app, available on Apple App Store and Google Play, helps patients navigate these challenges with personalized support.

Movement & Mental Wellbeing for Fibromyalgia

Gentle Movement

Low-impact activities like swimming and tai chi help reduce fibromyalgia pain and improve physical function without causing post-exertion malaise.

Mental Wellbeing

Cognitive behavioral therapy and mindfulness practices can help manage pain perception and reduce the emotional impact of fibromyalgia.

Sleep Hygiene

Quality sleep reduces fibromyalgia flares and improves energy levels, with consistent bedtime routines being particularly beneficial.

Anti-inflammatory Diet

Reducing processed foods and increasing omega-3 rich foods can help manage inflammation and reduce symptom severity in fibromyalgia patients.

Stress Management

Techniques like deep breathing and progressive muscle relaxation can help reduce stress-triggered pain flares common in fibromyalgia.

Hydration & Temperature

Staying well-hydrated and managing environmental temperature helps reduce muscle stiffness and prevent symptom exacerbation.

NICE or not NICE?

1. Background: NICE NG193 Overview

The NICE guidance on *Chronic pain (primary and secondary) in over 16s* was intended to move practice away from reliance on opioids and toward multidisciplinary, biopsychosocial approaches — emphasising CBT, exercise, and self-management.
However, it controversially:

- Excluded many pharmacological options (including most analgesics),
- Downplayed biomedical investigation, and
- Presented chronic pain largely as a *psychological or behavioural* issue.

2. Why Many Clinicians and Patients See It as Misogynistic

a. Gender Bias in "Medically Unexplained Symptoms"
Women are disproportionately diagnosed with "functional" or "primary" pain conditions (fibromyalgia, chronic fatigue, pelvic pain, migraine). Framing chronic pain as "pain without identifiable cause" echoes the historical pattern of psychologising women's suffering — from "hysteria" to "somatisation."

The guidance's wording (e.g., "pain without clear cause") risks reinforcing this bias, suggesting the problem lies in perception or coping rather than pathology.

b. Evidence Base Built on Male-Dominant Studies
Pharmacological evidence was downgraded partly because many pain trials historically under-represented women. Instead of correcting that imbalance, NICE effectively *penalised* conditions that affect women by declaring the evidence "insufficient" — leading to reduced access to treatments for them.

c. Disregard for Women's Testimony
Many women's pain reports (e.g., endometriosis, neuropathic pelvic pain) are dismissed as anxiety or catastrophising. The NICE framing around "reframing unhelpful thoughts" and "addressing coping behaviours" can feel invalidating — especially when women's experiences are already doubted in clinical settings.

I believe that NG 193 is deeply misogynistic, and I fear there is huge unconscious misogyny with ~~Pain~~ Medicine

Thank You

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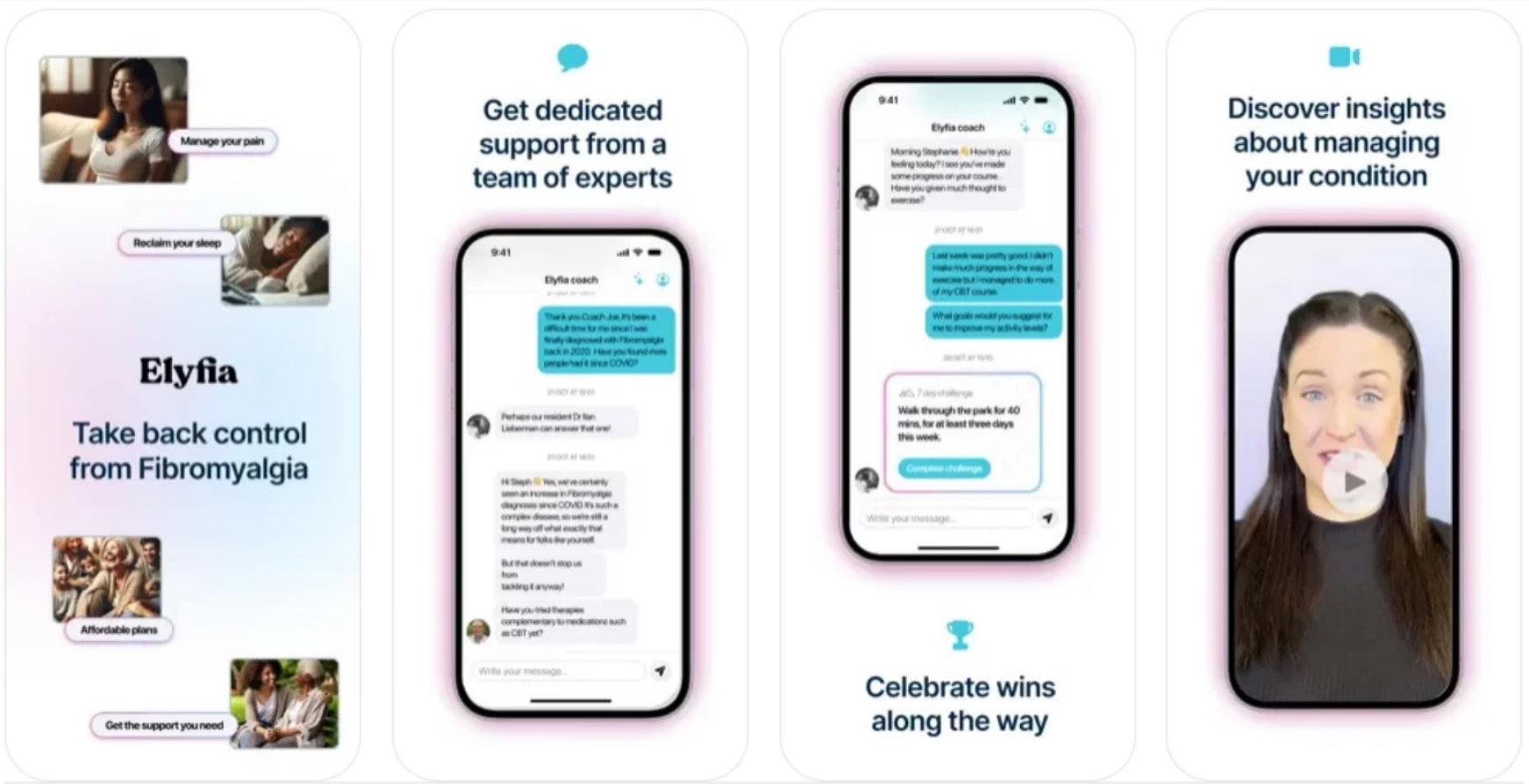
Elyfia.com Health Coaching for Fibromyalgia



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Elyfia helps fibromyalgia patients track and implement these beneficial activities through personalised recommendations tailored to their individual symptom patterns and triggers.

Download the Elyfia app today, available on the Apple App Store and Google Play Store for comprehensive fibromyalgia management support at your fingertips.

How Elyfia Delivers Results for Fibromyalgia

Assessment
Personalised evaluation of fibromyalgia symptoms, pain patterns, and environmental triggers.

Education
Evidence-based information on fibromyalgia management through chat and video resources.

Progress Tracking
Regular monitoring of pain levels, fatigue, cognitive symptoms, and treatment response.

Ongoing Support
Guidance through fibromyalgia flares with specialized coach accessibility.

Mood & Stress Management
Tools for emotional well-being, mindfulness techniques, and stress-reduction strategies to break the pain-stress cycle of fibromyalgia.

Our AI-enhanced platform enables fibromyalgia specialists to support 162 clients daily, making quality fibromyalgia care accessible and affordable.

Download Elyfia now, available on the Apple App Store and Google Play Store for comprehensive fibromyalgia management on the go.